THE RÔLE OF THE THEBESIAN VESSELS IN THE CIRCULATION OF THE HEART.

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PLATES 17 AND 18.

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Interest in the coronary circulation and blood vessels of the heart was very keen among the anatomists and physicians in the early part of the 18th century. They had begun to study the distribution of the coronary vessels and as a result of their dissections to construct theories of their function. Perfusion experiments were also carried out on human hearts obtained at necropsies and rapid advances were made in the knowledge of the coronary circulation. Text-books and anatomical works of the day devoted much space to the subject and clinicians began to correlate the postmortem with the clinical findings. The belief that special substances other than blood circulated in the blood vessels had begun to disappear.

This interest in the circulation of the heart walls was given a fresh stimulus by the publications of Raymond Vieussens in 1706 and by those of Adam Christian Thebesius 2 years later. Their discoveries of the channels connecting the coronary vessels directly with the chambers of the auricles and ventricles, introduced into the scheme of the circulation of the heart the vessels which are now generally known as Thebesian veins. In more recent times these vessels have received very little attention or study. Indeed, little is known of their anatomical relationships and no exact knowledge of the part they play in the coronary circulation exists. The importance, however, of a group of vessels which plays a part in the circulation of the heart itself, is obvious. It is the purpose of this paper to describe the previous work on the Thebesian vessels and their supposed functions and in addition, to present some observations made during the past 3 years

upon the relationship of these veins to the coronary arteries and upon some of their functions.

LITERATURE.

In 1706 Raymond Vieussens (1) published the results of a series of experiments which he had carried out upon human hearts after death together with others on beef and sheep hearts. In these experiments Vieussens ligated the vena cava above and below the right auricle of the heart and next ligated the pulmonary veins. Having thus blocked these outlets he injected a solution of safranine in alcohol into the coronary arteries. After the safranine had filled the tissues of the heart, he observed that it ran out, not only through the coronary sinus into the right auricle, but—and this is the point of the experiment—it also escaped directly into the cavities of the heart by way of small ducts in the walls of the auricles and ventricles. These openings he termed "ducti carnosi." He found a considerable number of them on the interior surfaces of the heart and after an extensive study concluded that they were continuous with the coronary arteries.

In 1708 Thebesius (2) published his description of the numerous openings for the venous blood in the auricles and ventricles. He identified the openings by injecting the water into the coronary sinus and observing its escape into the chambers. The same results were obtained by injecting air into the coronary veins, and on still other occasions he employed colored liquids mixed with wax and glue. Thebesius raised the question as to why the Creator had placed these small veins in the walls of the heart, and then very obligingly answered it by saying that they made possible a continuous blood flow in the heart by serving as an exit at the beginning of systole. After thus answering the question so satisfactorily to himself, he gave due credit and much praise to the Creator for being so foresighted as to anticipate the need and usefulness of these little vessels. Thebesius also stated that while he was writing his paper he was shown the report of Vieussens' experiments. Despite the fact, therefore, that the vessels bear the name of Thebesius, it is to Raymond Vieussens that credit for their discovery should go. He reported his findings first, but of even greater importance was his method of injection, namely, through the arteries. Thebesius injected the veins and both men described the same openings.

Philip Verheyen (3) in 1712 reviewed the work of Vieussens and repeated some of his experiments. He conceded that the experiments were very unusual, but added that Vieussens, like other great men, had drawn too many conclusions from them. Verheyen confirmed the presence of the openings in the walls of the heart cavities, but explained them as openings of coronary veins and denied rather emphatically that they were special ducts. Neither his experiments nor his arguments in favor of his claims were very convincing.

In 1715 Vieussens published a book (4) in which he confirmed his former discovery of the common openings and spoke of their distribution and function. It was in this delightful publication that he described in detail stenosis of the mitral

valve and the clinical and necropsy findings in a patient with insufficiency of the aortic valve.

Lancisi (5) repeated the experiments and confirmed the results of Thebesius. His explanation of the raison d'être of the Thebesian system is interesting even if not based upon experiment. Assuming that the heart beat was started by the rush of blood into the heart muscle, he thought a rapid and simultaneous emptying necessary in all parts of the heart. In the parts most distant from the right auricle the necessary exits were supplied by the Thebesian vessels, and by this means, he said all parts could be emptied simultaneously and as a result all parts could be filled instantly.

Sénac is quoted (6) as saying that injections of air, mercury or tallow pass into the ventricles of the heart directly and enter these cavities equally well whether poured into the coronary artery or coronary vein. Langer (7), Bochdalek (8) and many others have admitted the presence of these vessels and studied their distribution in the walls of the four chambers. Nussbaum (9) also studied the numbers and sites of the openings but concluded that they existed only in the auricles.

On the other side of the question, however, are arrayed Cruveilhier (10) and Theile (11) who denied that such vessels existed. In reading their views one is led to believe that they are opinions only, and not based upon original observation. Lannelongue (12) too, denied that the small openings in the heart walls were connected with the arteries and veins. He maintained that they were merely continuations of the numerous small canals that ran behind the plexus of pillars in the walls of the ventricles.

Haller has been quoted by several writers as denying the existence of Thebesian vessels but in one of his writings (13) he spoke of their presence and stated that their function was to return the blood of those deep seated coronary arteries which were unaccompanied by veins.

In more recent years Pratt (14) has demonstrated that an isolated cat heart can be kept beating for an hour by the perfusion of defibrinated blood through the Thebesian vessels. He believed the Thebesian veins were connected directly with the coronary veins and indirectly with the arteries through the capillaries. Pratt, moreover, explained the lack of infarcts in markedly sclerosed hearts by the presence of the Thebesian circulation.

The embryological development of the Thebesian veins has not been studied exhaustively but the few papers that exist indicate that these vessels are the remains of the primitive intertrabecular circulation of the embryo. Minot (15) found that the trabeculæ of the heart were made up in their earliest stage of young heart muscle cells covered with endothelium, but without capillaries of their own. It was his belief that the "sinusoidal" circulation of the embryonic heart was sufficient to nourish the trabecular muscle. Lewis (16) confirmed the work of Minot and followed the development of the sinusoids of the heart in the torpedo and rabbit and believed them to be the vessels that survive to some extent in the adult hearts, as the Thebesian veins.

Grant (17) has found that in the beginning the sinusoids were not connected with the coronary system, but grew into them at a later stage, as the coronary veins grew and branched in the myocardium. He (18) also described an anomaly of a child's heart in the ventricular muscle of which were large blood-filled spaces, communicating freely with the cavity of the ventricle as well as with the coronary vessels. They were interpreted as a persistence of the sinusoidal spaces of the embryonic heart.

In the hearts of certain fishes, turtles and frogs, the sinusoids persist in adult life, and it is through them that the inner surface of the heart wall is nourished. No capillaries have been found in the spongy surface supplied by the intertrabecular circulation.

Methods.

The observations which stimulated the work to be reported upon in this paper, were made during attempts to inject the capillaries of a

TABLE I.

Human Heart 17.

	1st measurement	2nd measurement
	cc.	*cc.*
Outflow from pulmonary artery	70	73
Outflow from aorta	190	190
Outflow from coronary sinus and veins	28	26
Leaks from cut edges of heart	60	52

human heart. The method which was employed for the injection was as follows:

About 48 hours after death a human heart was washed free of blood by perfusing salt solution (0.85 per cent) through the coronary arteries at a pressure of 160 mm. Hg. When the perfusate began to escape it was noted that the greater part of it ran out of the aorta and pulmonary artery and very little flowed from the coronary sinus. This observation was so startling that steps were taken immediately to measure the outflow from heart cavities and from the coronary sinus and other great veins. Glass cannulæ were introduced into the aorta and pulmonary artery in such manner that their ends extended well into the chambers of the ventricles. The coronary sinus and the posterior middle vein were also cannulated. At times several of the great veins emptied directly into the right auricle near the opening of the coronary sinus and just outside the valve of Vieussens. Whenever possible these veins were cannulated and the flow from them was

recorded. The coronary arteries were then perfused for 1 minute with normal salt solution at a pressure of 160 mm. Hg, and the perfusate was collected as it escaped from the various sources, and measured.

The results of such an experiment are shown in Table I.

Perfusion of salt solution (0.85 per cent) through the coronary arteries for 1 minute at 160 mm. Hg pressure.

Such results as those shown in Table I stimulated further study, and the experiment was repeated with the hearts obtained from a number of necropsies. Some of these were normal hearts, others were hypertrophied, while still others showed marked sclerosis of their vessels. The results of these experiments confirmed the findings in the earlier one (see Table II) namely, that 60–90 per cent of the coronary flow escaped through the Thebesian vessels directly into the chambers of the heart.

The possibility of rupture of some of the larger coronary vessels directly into the chambers or transudation of the salt solution into the cavities from the vessels was thought of as an explanation of the small amount of flow from the veins. Direct inspection, however, of the inner walls of the heart during perfusion with India ink showed the ink escaping only from the Thebesian vessels. Moreover, when the hearts were sectioned later, careful search failed to reveal any extravasation of the ink whatsoever. Further confirmation of the Thebesian vessels as the only source of the outflow from the chambers was furnished by perfusing with a suspension of gum acacia (6 per cent) in salt solution. This substance behaved in a manner similar to the saline perfusion, except that, as was to be expected, it ran more slowly (see Table II, Heart 43).

In several experiments agar and celloidin colored with dyes were perfused through the coronary arteries, and during the perfusion cold water or ice was introduced into the ventricles to harden these substances *in situ*. Subsequent sectioning of these hearts revealed small plugs of the injection mass protruding from the Thebesian openings. In one of the hearts in which celloidin was used as the injection mass seventeen openings of Thebesian vessels were found in the left ventricle with plugs of celloidin protruding from them (Fig. 1). The injection pressure used was 220 mm. of mercury and the mass was so thick

TABLE II.

Human heart No.	Age of patient	Necropsy diagnosis	Weight of heart	Perfusate used	Flow from left ventricle (Thebesian veins)	Flow from right ventricle (Thebesian veins)	Flow (Thebesian veins)	Flow from coronary sinus and vein	Leaks from cut surfaces	Pressure of perfusate	Amount of flow	Condition of coronary arteries
	yrs.		*gm.*		*cc.*	*cc.*	*per cent*	*cc.*	*cc.*	*mm. Hg*		
15	45	General peritonitis	320	Physiological salt solution .85 per cent	88 79	122 120	42 44	105 100	175 151	190	490 450	Normal
16	45	Chronic nephritis	500	Potassium ferrocyanide 3.2 per cent	75 70	125 125	76 73	20 27	40 45	155	260 267	Moderate arteriosclerosis
17	50	Acute purulent meningitis?	410	Physiological salt solution .85 per cent	176 190 190	65 70 73	73 76 91	25 28 26	 60 52	160	326 340 289	" "
20	50	Sarcomatosis	325	" "	235 240	250 260	78 74	50 80	80 95	220	615 675	" "
21	60	Chronic myocarditis; heart failure	300	" "	100 100	142 130	74 70	37 45	47 53	180	326 328	Slight sclerosis of arteries
22	51	Chronic nephritis	400	" "	175 175	235 215	74 74	20 15	120 120	150	550 525	Moderate sclerosis
24	42	Bronchopneumonia	220	" "	180 185	125 122	69 70	26 31	105 100	180	436 438	Normal

25	50	"	260	" "	0	60	68	4	23	180	87	Moderate arterio-
					1	62	69	5	23		91	sclerosis
26	65	Pulmonary tuber-	500	" "	183	200	84	10	60	150	453	" "
		culosis			176	208	83	8	70		462	
	mos.											
27	19	Bronchopneu-		" "	19	32	34	16	80	150	147	Normal
		monia										
	yrs.											
37	40	"	250	" "	0	100	23	30	300	150	430	Arteriosclerosis
					0	100	23	33	300		433	
43	43	Pulmonary tuber-	270	" "	140	215	72	60	75	150	490	Normal
		culosis			152	210	72	64	70		496	
				Acacia 6 per cent	175	175	58	75	170		595	
					168	163	59	65	160		556	
46	65	Cerebral hemor-	510	Physiological salt	232		62	56	85	150	373	Arteriosclerosis
		rhage		solution .85 per	246		64	60	75		381	
				cent								
47	82	Chronic nephritis	580	" "	210	300	76	35	125	150	670	"
		myocarditis			200	289	76	30	120		639	

These experiments were carried out as nearly as possible under exactly the same conditions. All injections were made approximately 48 hours post mortem. The temperature of the perfusate was kept at 37°C. and with the exception of Heart 43 all of the outflows were collected during 1 minute periods. In Heart 43 the salt solution was perfused for 2 minutes and the acacia for 5 minutes.

that it did not enter the capillaries. Indeed, only the larger vessels were injected. No openings were found in the auricles and only two were found in the right ventricle. These experiments, therefore, confirm the previous ones (Table II) for here again the capillaries were not filled. Finally microscopic examination of sections from numerous parts of the hearts showed that the dyes and injection material had remained within the walls of the vessels.

Crainicianu (19), in perfusing hearts, noted this large escape from the ventricles but did not pursue it further. In 1913 Starling and Evans (20) during a study of the metabolism of the heart and lungs noted that about 40 per cent of the coronary blood did not return *via* the coronary sinus. They did not state, however, whether they took into consideration the other great veins which empty directly into the right auricle or the Thebesian flow to the left auricle and ventricle.

On gross inspection of the hearts used in the experiments of Table II it was obvious that the injection was uneven and incomplete. The larger vessels were well filled with ink or dye, but for the most part the heart was not colored by the injection material. Sections from numerous points in the walls of all the chambers were prepared and a careful microscopic study of these brought out the surprising fact that in most instances scarcely any of the capillaries were injected. The large arteries and veins were everywhere well filled or stained with the injection mass, but in the great majority of the microscopic fields very few if any capillaries could be found (Fig. 2). In certain fields small groups of capillaries were filled, but in no instance in the study of fifty human hearts was the capillary bed entirely injected. Even in the hearts in which some of the capillaries were injected they were so few in number that it was obviously impossible for them to take care of the profuse flow from the arteries into the cavities of the auricles and ventricles. In two hearts there was a scant flow through the Thebesian vessels, and in these several areas showed almost completely filled capillaries. The injection, however, was uneven and several large spots in the walls remained uninjected.

Here then is evidence that a direct connection exists between the coronary arteries and the chambers of the heart. The fact that so great a flow between the two can take place without going through the

capillaries shows the connecting channels to be of considerable size. The nature of this connection has not been definitely determined. It may possibly be a direct one from artery to Thebesian vessel, or more probably it may go from an artery into a large vein and thence into the heart cavity. Two of the smaller Thebesian vessels have been traced by serial sections, and their branchings studied. For convenience the smaller papillary muscles were selected because in so small a structure the course of the vessel would necessarily be a short one. In the two vessels so studied large venous branches were given off immediately beneath the endocardium and these branches divided immediately into venules and capillaries as illustrated in Fig. 3. Some of the Thebesian veins, therefore, drain the capillaries. This outlet for the capillary blood obviates the longer route of return through the veins to the coronary sinus and right auricle. The study has not progressed far enough to determine the relative number of Thebesian veins that drain capillaries as compared to those that connect directly with larger veins or through them with arteries. It is clear, however, that three types of connections exist, (1) the direct connection between the arteries and the Thebesian vessels, as shown by the celloidin injections, (2) the venous connection with the Thebesian vessels and (3) the capillaries which run directly into the Thebesian vessels.

The distribution of the Thebesian openings varied greatly but they were usually most numerous in the walls of the ventricles, in the pocket of the apex and in and around the bases of the papillary muscles. In the hearts studied there were relatively few openings into the auricles, and in two instances it was not possible to demonstrate any openings at all in either auricle. This was determined by measuring the flow from each chamber separately. The following protocol is illustrative.

Protocol.

Heart 47.—Male. Age 82. Chronic myocarditis with heart failure. A human heart, 24 hours post mortem, had cannulæ placed in the pulmonary artery, the coronary sinus and posterior middle vein, in the aorta and in the coronary arteries. The mitral valve was sewed together very tightly, as was also the tricuspid valve, in such fashion that they were water tight. A cannula was then sewed into each auricle. Following this, the coronary arteries were perfused at a pressure of 150

mm. of mercury, and flows from the various chambers were recorded for 1 minute periods. They were as follows:

	1st measurement	2nd measurement
	cc.	*cc.*
Pulmonary artery	300	289
Aorta	210	200
Coronary sinus and vein	35	30
Right auricle	0	0
Left auricle	0	0
Leaks	125	120

Following these measurements Berlin blue was injected into the coronary arteries at a pressure of 150 mm., and simultaneously carmine solution was injected through the coronary sinus and the posterior middle vein. The injection pressure continued for a few minutes following which the heart was opened and examined. It was interesting to note that most of the blue had emerged near the apex in each ventricle. The red was not so well localized. Neither red nor blue appeared in the auricles.

The number and size of Thebesian vessels in the various chambers also varied greatly. At times the flow was much greater from the right side while at others greater from the left, but the average from all the hearts showed a slightly greater flow from the right. The difference, however, was not significant.

Changing the perfusion pressure did not change the relative proportion of flow from the two sides of the heart, but did affect the rate of flow. Nor did the temperature of the heart influence the flow, for perfusions were carried out at various temperatures without effect upon the amount. It might be stated, however, that with few exceptions all the hearts were kept from 24–48 hours in the ice box, and then gently massaged and brought to a temperature of 45°C. before being perfused.

Attempts to inject the capillaries of a heart while in rigor mortis met with no better success. Nor did the use of this method with fresh warm hearts obtained soon after death give any better results. In still other hearts potassium sulfocyanate (10 per cent) was perfused through the vessels for 1 hour before they were injected with dye, as this procedure had been found to relax the greater vessels (21). These

hearts showed no better injections of the capillaries than did the others injected by the same method, in the absence of potassium sulfocyanate.

Vieussens, in his studies of the ducts opening into the heart chambers, made his injections into the coronary arteries, while Thebesius introduced his blowpipe into the coronary vein and observed the bubbles escaping into the immersed chambers. In this study the experiments of both Vieussens and Thebesius have been confirmed. Moreover, when perfusion was carried out through the coronary sinus at pressures ranging from 50–150 mm. Hg, almost all of the perfusate ran out through the Thebesian vessels, and in only a few instances did a few drops escape through the coronary arteries. This result was obtained when saline, acacia, agar and gelatin were used. Again in these hearts the capillaries were not injected, thus showing that the perfusate must have escaped by a more direct and larger communication between the veins and the Thebesian vessels, without having passed through the capillaries.

Perfusion was next carried out through the Thebesian vessels. Cannulæ were introduced into both coronary arteries, the coronary sinus and the other great veins which did not empty into the coronary sinus. All other outlets to the chambers were then carefully tied off after the chambers had been filled with 2 per cent Berlin blue in salt solution. Compressing the heart caused most of the fluid to run out of the veins, a few drops only escaping through the arteries. Histological sections of these hearts also showed little dye in the capillaries. The veins and a few arteries, however, were filled with dye.

These experiments have shown the existence of connections (1) between the arteries and Thebesian vessels and (2) between the veins and Thebesian vessels. In neither instance is the junction made through the capillaries alone. Before discussing the function of these channels it will be of interest to report the results of further experiments.

It will be noted that in all the experiments just described capillaries were rarely filled with the perfusing dye, and in no instance was more than a small fraction of the total number of capillaries injected. This held true even in the hearts where perfusion pressures were raised sufficiently to rupture the arteries.

It was routine practise in these experiments to wash the hearts free

of blood by perfusing them with saline before adding the dye to the perfusate. This procedure invariably distended the walls and dilated the chambers of the non-elastic dead heart. The mere perfusion distended it gradually and the fluid escaping from the Thebesian vessels stretched the walls as the cavities filled. Heyde (22) had shown that slight dilatation of the heart walls resulted in a slowing of capillary flow. These experiments confirmed that finding and failed to fill the capillaries while the heart walls were stretched.

In view of these findings the dye was introduced directly without previous washing out with salt solution in the manner described in the following experiment.

Protocol.

Heart 44.—This heart was obtained 2 hours post mortem and was still warm. Cannulæ were placed in the coronary arteries as quickly as possible and the heart, while the chambers were still collapsed and there was no stretching whatever, was injected with India ink diluted with an equal part of distilled water, heated to 45° at a pressure of 220 mm. of mercury.

The whole heart and aorta immediately became very black and the injection appeared excellent. Special care was taken to get the injection material in at a high pressure suddenly, to avoid distention of the walls of the heart. This is distinctly in contrast to the earlier human heart injections where the hearts were first perfused with salt solution in order to wash out the blood, and in which procedure the chambers were greatly distended. Later perfusion of hearts so injected with dye or ink invariably injected the Thebesian system and very few capillaries.

In this experiment and in all others carried out in the same manner a large number of the capillaries was injected, sometimes as many as 65 to 85 per cent of the total number. In several instances the injection was practically complete in certain areas of the muscle while in other parts the capillaries were only partially filled. It is of great import that the flow from the Thebesian vessels was very small in the hearts that showed such good capillary injection. In the earlier experiments where the coronary arteries were perfused with salt solution before the dye was introduced, it was noted that the capillaries were free from blood shortly after the perfusion was begun. The color of muscle changed from red to pale pink. Hence, when the dye or ink was injected without previous perfusion, it ran into the capillaries

until the heart became distended and then escaped through the Thebesian vessels into the cavities. This seemed the most plausible explanation of the results obtained. On this basis, therefore, the next step in the investigation was clearly indicated, namely, to study the flow from the Thebesian vessels in a beating heart. Consequently, human hearts obtained within 2 or 3 hours post mortem were perfused with oxygenated Locke-Rosenheim (23) solution. Usually they began to beat within a few minutes after the perfusion was started. The following experiment illustrates the method used.

Heart 52.—July 15, 1926. This heart was obtained 2 hours post mortem from the autopsy of boy, age 15, who died from Hodgkin's disease. Heart normal in size. Both coronary arteries were cannulated and a large cannula was tied into the coronary sinus. During this preparation the heart was kept at 37°C. in salt solution. 2 hours and 30 minutes postmortem perfusion of the coronary arteries was started with oxygenated Locke-Rosenheim solution. The temperature of the perfusion fluid was constant at 37°C. and the perfusion pressure was kept at 50–55 cm. of water. Within 5 minutes the auricles began to show very feeble beats and these were followed immediately by beats of both ventricles. A small dose of adrenalin (0.1 cc. of 1:1000) was injected very slowly into the perfusing fluid, and following this the auricles and ventricles began to beat strongly and normally, so that large spurts of fluid were forced from the aorta and the pulmonary artery. The rhythm became regular and the rate became constant at 62 beats per minute. The following flows were then recorded for 1 minute intervals.

After these flows were recorded a suspension of Berlin blue (2 per cent) in salt solution was injected into the perfusion cannulæ in the coronary arteries at sufficient rate to replace the Locke-Rosenheim solution. The pressure remained at 50 cm. water. The heart immediately turned to a deep blue color and glacial acetic acid was run into the coronary arteries. The acid stopped the heart beats instantly.

Similar results were obtained in another experiment, the single difference being that the left ventricle remained in systole and did not beat while the other three chambers were beating regularly. The outflow was measured during 1 minute before and after the heart began to beat.

Perfusion of the coronary arteries with Locke-Rosenheim solution at 50 cm. water pressure 37°C. 3 hours post mortem. The first flow was recorded before the heart began to beat. In this heart the left ventricle did not beat.

When the figures of Tables III and IV are contrasted with those in Tables I and II, the large outflow from the coronary sinus in Tables III and IV is very impressive, and all the more so when it is realized that the outflow from the other great veins not only was not included under the flow from the coronary sinus but was added to the ventricular outflow. In many of the experiments on dead hearts the outflow from the other great veins of the heart equalled that of the coronary sinus. The second column of figures, therefore, contains

TABLE III.

Heart 52.

	From the coronary sinus	From the other veins, Thebesian vessels and leaks	Heart rate per min.
	cc.	*cc.*	
1st measurement	155	295	62
2nd measurement	115	225	62

TABLE IV.

Heart. 41. Male. Age 75. Death from Lobar Pneumonia. Heart Normal for Age.

	From the coronary sinus	From the other veins, Thebesian vessels and leaks	Heart rate per min.
	cc.	*cc.*	
1st measurement	205	360	Not beating
2nd measurement	215	330	86
3rd measurement	125	195	100

part of the venous outflow as well as the Thebesian flow and that part of the perfusate which escaped by leaking from the cut edges. This last source alone—the leaks—averaged much more in Table II than the flow from the coronary sinus and the veins. Of equal importance, too, in this heart was a fact brought out upon microscopic study of the sections from all parts of the muscle. The capillaries were completely and evenly injected (Fig. 4). Thus, in a beating heart, in the absence of dilatation, the capillaries were filled and the Thebesian flow was greatly diminished. The left ventricle which did not beat, was in

rigor and its capillaries were very imperfectly injected in contrast to those of the other chambers which were beating.

The results of these experiments were confirmed by another which differed only in the fact that flows were recorded before and after the heart began to beat. The protocol follows.

Human Heart 53.—Weight 300 gm. Male. Age 52. Death from pernicious anemia. This heart was obtained about 2½ hours post mortem. Cannulæ were introduced into both coronary arteries and into the coronary sinus, the latter for recording outflow. At about 3 hours postmortem perfusion with oxygenated Locke-Rosenheim solution was started through the coronary arteries. The temperature of the perfusion fluid was kept at 37°C. at the point of entrance into the arteries. The perfusion pressure was 55 cm. water. As the perfusion was started the chambers filled gradually and there was a very slight dilatation of the right ventricle, which increased gradually until the heart began to beat. Before any beats occurred the outflow was measured during two periods of 2 minutes each. They were as follows:

	Outflow from coronary sinus	Outflow from other veins, Thebesian vessels and leaks
	cc.	*cc.*
1st measurement	210	525
2nd measurement	190	510

After perfusing about 5 minutes 0.5 cc. adrenalin (1:10,000) was added very slowly to the perfusing fluid. Following this the right auricle began to twitch and immediately both auricles began to beat regularly. Next the right ventricle began to beat and finally the left ventricle joined in and beat very feebly. After another 5 minutes the rhythm was regular and the rate was constant at 78 per minute. At this point the flow was recorded for two more periods of 1 minute each, the results of which follow:

	Outflow from coronary sinus	Outflow from other veins, Thebesian vessels and leaks	Rate per min.
	cc.	*cc.*	
1st measurement	260	550	78
2nd measurement	268	545	78

Before further flows could be recorded the beat of the left ventricle became very weak and finally lost all force so that it merely twitched. At this point Berlin blue 2 per cent was introduced into the coronary artery cannulæ at such rate as

to replace the Locke-Rosenheim solution, and the beat of the heart immediately became weaker. Glacial acetic acid was then added to the perfusate and the heart beat ceased. Sections were cut from various parts of the walls, measured and placed in 10 per cent formalin (aqueous) for fixation.

The results of this experiment add to the value of the figures of the previous one (Heart 52) and in addition show a very slight increase of the venous outflow in the beating heart over the outflow before it began to beat. This heart also showed a good capillary injection except in the left ventricle which had stopped beating before the dye was injected.

The important point of this experiment is the relative change in the amount of outflow from the different sources when contrasted to the perfusion of dead hearts. In the beating heart (Heart 52) it did not

TABLE V.

Heart 52 Beating. Heart 24 Not Beating.

	Outflow from Thebesian vessels*	Outflow from coronary sinus and veins	Leaks
	cc.	*cc.*	*cc.*
Heart 52	140	180	75
Heart 24	305	28	102

* Average of combined outflow from the two ventricles.

seem wise to spend the time required in introducing cannulæ into the pulmonary artery, aorta and great veins emptying directly into the right auricle. Consequently, the outflow from the chambers was mixed with leakage from the cut surfaces, and in addition contained the outflow of all the veins emptying into the right auricle except that from the coronary sinus. In many of the perfusion experiments on dead hearts the outflows from the coronary sinus and from certain other great veins were measured separately and the average outflow from the latter was a third to a half of that from the coronary sinus. If one takes that figure as the flow from the other veins and assumes that the leakage was 75 cc. the following values can be calculated for the flows from the different sources.

When these calculations have been made by subtracting the leaks

and venous flow (other than from the coronary sinus) from collected outflow and adding the venous flow to that of the coronary sinus a figure is found which is quite in contrast to those found on perfusing dead hearts.

The evidence thus far considered has been obtained from hearts after death or from isolated beating hearts. In support of this evidence the results of thirty-five experiments on intact cats are offered. The cats were carefully anesthetized with sodium barbital and when completely under the anesthetic, the chest was opened under artificial respiration in such manner that the heart was exposed. A dye or India ink[1] was injected into the left auricle or left ventricle and the heart, by pumping the dye or India ink into the coronary arteries, injected itself. Immediately after the injection of India ink was started, the arteries first filled and appeared as black stripes on the heart. As the ink reached and filled the capillaries the entire heart became uniformly black but remained so only for a brief second or more when it suddenly began to dilate. During the dilatation, even though the ink injection was continuous, the beats became very feeble and the color changed from deep black to a pale pinkish color, as the feeble beats "milked" or squeezed the dye from the capillaries.

An explanation of the change in the color of the heart as it dilated and began to beat feebly was difficult, but it was furnished by the two experiments which follow.

Cat 2.—Weight of cat 3 kilos. May 14, 1924. Anesthesia was induced by means of sodium barbital (0.6 gm. per kilo intraperitoneally). Blood pressure was then registered by means of a cannula in the carotid artery. The chest was opened under artificial respiration and the pericardium incised in such a manner that the left auricle and ventricle came fully into view. India ink diluted with an equal part of distilled water at a temperature of 37°C. was then injected directly into the left ventricle. As the injection began the aorta was clamped at the level of the right carotid artery. The pressure used in injecting the ink was barely sufficient to cause the ink to enter the ventricle against ventricular pressure. When the heart began to turn black, showing a capillary injection, 5 mg. of histamine was injected along with the ink. The total amount of ink used was 20 cc. At a certain time during the injection the heart turned very black and at this

[1] Berlin blue 2 per cent suspension in normal saline. Either Weber's or Higgins' India ink was used, as a 50 per cent suspension in distilled water. Both dyes and inks were filtered immediately before using.

point the large clamp was placed on the heart in the auriculoventricular groove. The vessels at the base were also clamped, the heart excised and placed in 10 per cent formalin with the clamps in place.

This heart ceased to beat almost immediately after the clamp was placed in the auriculoventricular groove. The injection of the capillaries appeared to be perfect on gross examination.

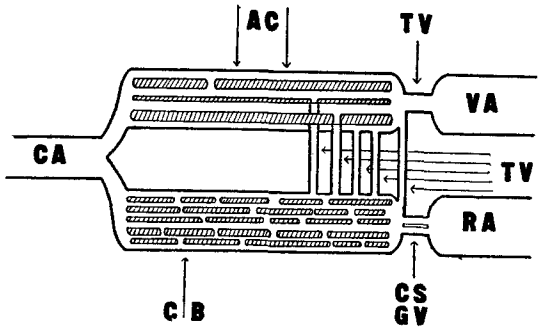
Cat 5.—Weight 3.1 kilos. May 20, 1924. Anesthesia was induced by means of sodium barbital (0.5 gm. per kilo intraperitoneally). Both carotid arteries were cannulated as was also the femoral vein. Blood pressure tracings were made from the left carotid and finally the abdomen was opened and a ligature placed around the aorta but it was not tied. Berlin blue (2 per cent suspension in 0.85 per cent salt solution) was slowly injected into the femoral vein. After approximately 10 cc. had been injected the blood pressure began to fall and the injection was made more rapidly from that time on, but the rate was never such as to raise the blood pressure above the original level.

The chest was hurriedly opened and it was found that the heart had stopped beating in systole and was deep blue in color. Additional Berlin blue was then injected through the right carotid artery after the ligature around the aorta had been tied. The injection pressure was 220 mm. of mercury. 10 per cent formalin was finally run in through the same artery at the same pressure. The chest was opened, the vessels of the heart clamped at the base, the heart excised and placed in 10 per cent formalin.

The important point that was common to these two experiments was the fact that the heart ceased to beat in each instance while the capillaries were filled and before it began to dilate. Furthermore, microscopic sections of both hearts proved the capillaries to be perfectly injected. The explanation to be given of this behavior, while not necessarily a final one, seems entirely reasonable. As the injection of ink was begun it entered the capillaries and cutting off the oxygen to the muscle, caused the heart to dilate. As dilatation occurred the capillaries were stretched and thereby further entrance of ink into them was prevented. But the feeble beats of the dilated heart were sufficient to squeeze the ink out of the capillaries. Such circulation as continued was almost certainly through the Thebesian vessels which were found well injected with the ink when the sections were studied with the microscope. Repeated efforts have been made to duplicate these experiments following each step with the greatest care, but all have failed because it has not been possible to stop the hearts at the proper stage during the injection, while the capillaries were filled. Sections in these later instances showed that only the large vessels were filled.

After witnessing these experiments one can hardly refrain from speculation as to the reason why the cardiac capillaries are filled with such difficulty and what part, if any, the Thebesian vessels play as an impediment to their filling. The results just reported furnish data which will serve as a basis for an hypothesis.

The facts that capillaries are perfectly filled only in a beating heart, that in these experiments at least, they fill less completely in a non-distended dead heart, and scarcely at all in a dilated heart, suggest the Thebesian vessels as a cause for the lack of success in the last two instances. The beating heart therefore must at some stage of its cycle close the Thebesian vessels and force the blood through the capillaries. This is not at all unreasonable when one considers the pressure relations in the left ventricle and the aorta during systole and diastole. If one accepts for the moment the presence of the direct communications between the



TEXT-FIG. 1. *C A* = coronary arteries. *A C* = arterial communications with Thebesian vessels. *T V* = Thebesian vessels. *V A* = cavities of ventricles and auricles. *C B* = capillary bed. *C S* = coronary sinus. *G V* = great veins emptying directly into the right auricle. *R A* = right auricle.

coronary arteries and the heart chambers on one hand and between the veins and chambers on the other, a chart can be drawn which illustrates diagrammatically the various vascular channels of the heart. The commonly accepted route of blood flow in the heart is from the aorta into the coronary arteries and thence through the capillaries into the large veins which empty into the right auricle directly or *via* the coronary sinus. In the diagram in Text-fig. 1, this route would be shown by having the blood flow from the coronary arteries (*C A*) through the capillary bed (*C B*), and the coronary sinus and veins (*C S* and *G V*) into the right auricle (*R A*). The experiments just reported have shown a direct communication between the coronary arteries (*C A*) and the heart chambers *via* the channels (*A C*) to the Thebesian vessels (*T V*). And in dilated dead hearts these channels

proved so readily accessible that approximately 60–90 per cent of the fluid perfused into the coronary arteries escaped through them. In beating hearts the amount was considerably less and more perfusate ran through the capillaries (*C B*) into the coronary sinus and veins (*C S* and *G V*). If the pressure that exists in life during the various phases of the cardiac cycle be considered, a hint of the use of the Thebesian vessels may be found. During ventricular systole the pressure is the same, of course, in the left ventricle and at the mouth of the coronary arteries, but the pressure in the right ventricle and in both auricles is less than it is in the coronary arteries. In diastole, however, with the aortic valves closed, the pressure in the coronary arteries (*C A*) is greater than in any of the heart chambers, hence an increased outflow from the Thebesian vessels (*T V*) into the heart chambers (*V A*) would be expected. Even during ventricular systole there is possibly a little outflow into the right ventricle, and an unhindered flow into the auricles. The number of openings in the auricles, however, is usually small.

This set of pressures would certainly retard the flow from the coronary arteries (*C A*) to the right ventricle and stop it from the coronary arteries (*C A*) to the left ventricle; and would, therefore, greatly inhibit the flow from the coronary arteries (*C A*) to the chambers (*V A*) during systole. The alternate route of flow would be through the capillary bed (*C B*). The Thebesian system, therefore, would serve as an additional means of escape of the blood in the heart wall and thereby enable the coronary vessels to empty themselves very rapidly. The veins also communicate directly with the Thebesian vessels, therefore the latter might serve as a means of rapid emptying of the entire vascular bed of the heart during diastole. Moreover, since some of the Thebesian vessels are connected directly with the capillaries, it may be possible for blood to flow from the ventricle through these vessels into the capillaries.

Such speculations as have been discussed are interesting but extremely difficult of proof. They are offered as possible explanations with full realization that further work is indicated which may or may not support them. So long as they are recognized as theory rather than fact they may serve to stimulate further study of the function of these vessels in normal hearts.

Finally another very important function of the Thebesian vessels has been demonstrated by the findings in two hearts at the post-mortem table.[2] This function has been proved more conclusively by the clinical histories and necropsy findings in these two cases than it could be by any experiment in the laboratory. In each of the hearts

[2] It was possible to study and use these hearts through the kindness of Dr. Timothy Leary. The cases are being reported in detail in another paper.

there was complete closure of both orifices of the coronary arteries. The first one was found in a negro woman, about 26 years of age, who had worked and earned her living as a seamstress up to within a few days of her death. She was found dead in her room and as she lived alone the nature of her death could not be learned. A syphilitic process in the aorta, probably of long standing, had completely closed and obliterated the orifices of both coronary arteries.

The second heart was from a man 35 years old who at the time of his death was working as a seaman. It was learned that he had been considered very lazy by other members of the crew but he was able to do his work and so far as they knew, did it without any symptoms or signs of heart disease. Between his jobs as a seaman he worked for an awning company and assisted in putting up and removing awnings from houses. In this heart also there was a definite syphilitic aortitis which had closed the orifices of both coronary arteries.

Needless to say, in both instances a very careful search for other openings of the arteries was made, but without success. Attempts to inject fluid and pass probes through the orifices also failed. Moreover, sections through the vessels $\frac{1}{2}$ cm. outside the aorta revealed normal lumens. The orifices had, therefore, been closed by the disease in the aorta, and the closure almost certainly had been a very gradual one. Sections through the arteries at their orifices showed complete closure in all vessels and in these the process was of long standing.

In each case a heart without openings for the coronary arteries had maintained an efficient circulation which enabled the person to earn a living. The obvious questions are where and how did these hearts get sufficient circulation to function so efficiently? The answer is not difficult for the only other entrance to the coronary circulation is through the Thebesian vessels and in the instances just cited, at least, they were able to supply the heart muscle with the necessary blood to maintain a wage-earning life. It should be emphasized at this point that the closure of the coronary orifices in these two cases was a gradual one. It was this time element, most probably, that enabled the Thebesian circulation to take over the new duties and perform them so efficiently. One does not need to search far afield for evidence to support this claim. It is commonplace to find at necropsies extensive

sclerosis of the coronary arteries, so well advanced that it is difficult to introduce the point of a pin into the calcified lumens. In many instances these people had been comparatively well and healthy individuals, with dyspnea on exertion perhaps, but falling into the group of men considered normal at their age. In such conditions it is certain that the process of occlusion was a gradual one. Their clinical histories stand out in sharp contrast to those of patients with sudden closure of one branch of a coronary artery. Many of these die at the time of the closure while others recover after acute heart failure. In certain emergencies, therefore, particularly when allowed sufficient time to adapt themselves, the Thebesian vessels can take over the function of the coronary arteries.

SUMMARY.

In summary, evidence has been presented to show a direct connection other than through the capillaries between the coronary arteries and the chambers of the heart.

This connection was shown by perfusion, injections and serial sections to be through the Thebesian veins. Communications between the larger coronary veins and the Thebesian veins were also demonstrated by the same methods.

Serial sections through Thebesian veins have shown capillaries draining directly into them. Under certain conditions it has been shown that as much as 90 per cent of the arterial flow may escape *via* the Thebesian vessels.

Lastly, in the event of gradual closure of the orifices of the coronary arteries, the Thebesian vessels can supply the heart muscle with sufficient blood to enable it to maintain an efficient circulation.

This work has been in progress for 4 years during which time Dr. William B. Stevens assisted in many of the earlier experiments. I wish to express my deep appreciation to Misses Olivia Ames and Sylvia Warren, and Drs. Henry Jackson, Jr., and Joseph M. Hayman, Jr., for their assistance in translating the older French and Latin papers; and to Dr. Francis W. Peabody for his very helpful criticisms and advice throughout the investigation.

BIBLIOGRAPHY.

1. Vieussens, R., Nouvelle decouvertes sur le coeur, Toulouse, 1706.
2. Thebesius, A. C., Dissertatio medica de circulo sanguinis in corde, Lugduni Batavorum, 1708.
3. Verheyen, P., Anatomiæ corporis humani, Coloniæ, 1712.
4. Vieussens, R., Traité nouveau de la structure et des causes de mouvement naturel du coeur, Toulouse, 1715.
5. Lancisi, J. M., A secretiori cubiculo et Archiatri Pontificii, de motu cordis, et aneurysmatibus (opus posthumum), Lugduni Batavorum, 1740.
6. Sénac, J. B., Traité de la structure du coeur, Paris, 1749, quoted by Lannelongue (12).
7. Langer, L., Die foramina Thebesii in Herzen des Menschen, *Sitzungsber. k. Akad. Wissensch., Math.-naturw. Cl., Wien*, 1880, 3 Abt., lxxxii, 25.
8. Bochdalek, V. A., *Arch. Anat.*, 1868, 302.
9. Nussbaum, A., Über das Gefäszsystem des Herzens, *Arch. mikr. Anat.*, 1912, lxxx, 450.
10. Cruveilhier, J., Anatomie descriptive, Paris, 1834, iii, 1.
11. Theile, F. G., Myologie et angéiologie, in Encyclopédie anatomique, 1843, iii, 586.
12. Lannelongue, O., Circulation veineuse des parois auriculaires du coeur, Paris, 1867.
13. Haller, A., First lines of physiology, English translation, Edinburgh, 1786, i, 75.
14. Pratt, F. H., *Am. J. Physiol.*, 1898, i, 86.
15. Minot, C. S., *Proc. Boston Soc. Natural Hist.*, 1900, xxix, 185.
16. Lewis, F. T., *Anat. Anz.*, 1904, xxv, 261.
17. Grant, R. T., *Heart*, 1926, xiii, 261.
18. Grant, R. T., *Heart*, 1926, xiii, 273.
19. Crainicianu, A., *Virchows Arch. path. Anat.*, 1922, ccxxxviii, 1.
20. Evans, C. L., and Starling, E. H., *J. Physiol.*, 1913, xlvi, 413.
21. McCordick, quoted by Gross, L., The blood supply to the Heart, New York, 1921.
22. Heyde, I. M., *Am. J. Physiol.*, 1898, i, 215.
23. Locke, F. S., and Rosenheim, O., *J. Physiol.*, 1907–08, xxxvi, 205.

EXPLANATION OF PLATES.

PLATE 17.

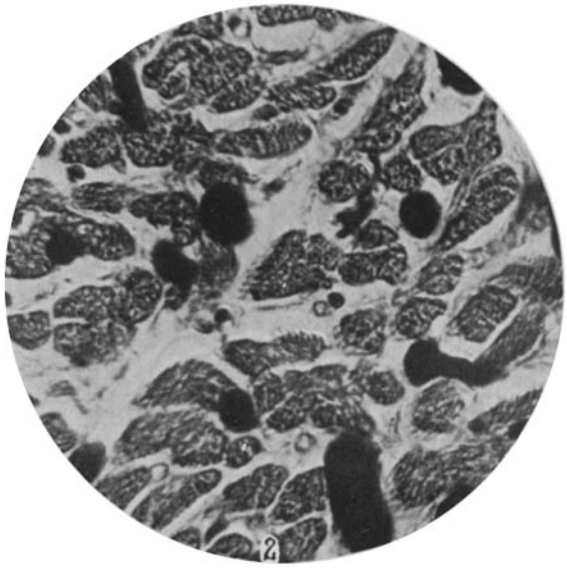
FIG. 1. Left ventricle of heart injected with celloidin, showing the celloidin plugs protruding from the Thebesian vessels. The plugs have been covered with white ink in order to make them show more distinctly in the photograph.

FIG. 2. Cross-section of muscle fibres from the left ventricle of Human Heart 14 showing the larger vessels well filled, but scarcely any capillary injection.

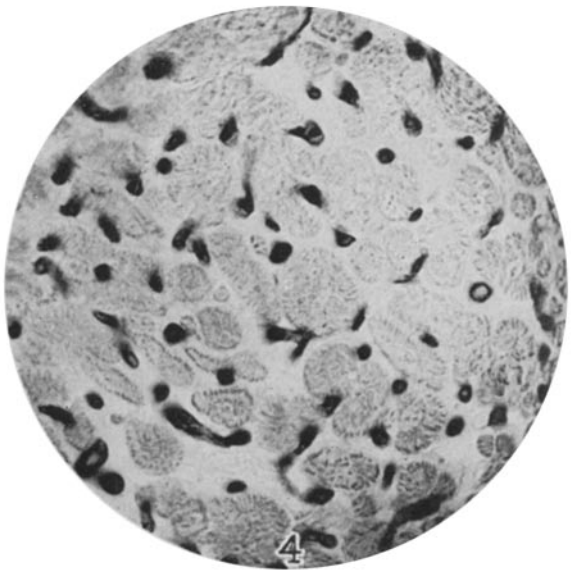
PLATE 18.

FIG. 3. One of a series of sections through a Thebesian vein (the channel running across the middle of the field) showing a vein and capillaries emptying into the Thebesian vein. This section lay less than 0.5 mm. below the endocardial surface.

FIG. 4. Cross-section of muscle fibres and capillaries of right ventricle of Human Heart 41 injected while beating.



(Wearn: Thebesian vessels in heart circulation.)



(Wearn: Thebesian vessels in heart circulation.)